Neurotoxic A1 astrocytes, which secrete toxic factors that kill mature oligodendrocytes and neurons, have been shown to be involved in a variety of neurodegenerative diseases and spinal cord injuries. The detrimental role of A1 astrocytes in traumatic brain injury has also been well documented.

Results showed a heterogeneous population of A1 and A2 reactive astrocytes on the shunts with obstructed shunts having a significantly higher proportion of A2 astrocytes compared to non-obstructed shunts. In addition, the pro-A2 cytokine IL-6 inducing proliferation of astrocytes was found at higher concentrations among CSF from obstructed samples. Consequently, in the in vitro model of astrocyte growth on shunts, cytokine neutralizing antibodies were used to prevent activation of resting astrocytes into the A1 and A2 phenotypes which resulted in a significant reduction in both A1 and A2 growth.

Therefore, targeting cytokines involved with astrocyte A1 and A2 activation is a promising intervention aimed to prevent shunt obstruction [1].

1)
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